Blood gas analysis for bedside diagnosis

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····· ABSTRACT

Arterial blood gas is an important routine investigation to monitor the acid-base balance of patients, effectiveness of gas exchange, and the state of their voluntary respiratory control. Majority of the oral and maxillofacial surgeons find it difficult to interpret and clinically correlate the arterial blood gas report in their everyday practice. This has led to underutilization of this simple tool. The present article aims to simplify arterial blood gas analysis for a rapid and easy bedside interpretation. In context of oral and maxillofacial surgery, arterial blood gas analysis plays a vital role in the monitoring of postoperative patients, patients receiving oxygen therapy, those on intensive support, or with maxillofacial trauma with significant blood loss, sepsis, and comorbid conditions like diabetes, kidney disorders, Cardiovascular system (CVS) conditions, and so on. The value of this analysis is limited by the understanding of the basic physiology and ability of the surgeon to interpret the report. Using a systematic and logical approach by using these steps would make the interpretation simple and easy to use for oral and maxillofacial surgeons.

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INTRODUCTION

Arterial blood gas analysis is an important routine investigation to monitor the acid-base balance of patients, effectiveness of gas exchange, and the state of their voluntary respiratory control.^[1]

In context of oral and maxillofacial surgery, arterial blood gas analysis plays a vital role in monitoring of postoperative patients, patients receiving oxygen therapy, those on intensive support, or with maxillofacial trauma with significant blood loss, sepsis, and comorbid conditions like diabetes, kidney disorders, Cardiovascular system (CVS) conditions, and so on.

Considering the spectrum of its uses, this useful and simple tool has been underutilized, most frequently due to the difficulty in proper understanding, interpretation, and application in management.

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This article aims to simplify the arterial blood gas analysis for a rapid and easy bedside interpretation for oral and maxillofacial surgeons.

The basic physiology

Our body functions in a relatively narrow alkaline environment (pH: 7.35-7.45). Maintenance of normal physiologic function is closely related to the maintenance of pH within this range.^[2]

The two main mechanisms for this balance are respiratory and metabolic. [2]

The normal blood pH range is 7.35-7.45.

- If pH <7.35, the blood is said to be acidic.
- If pH >7.45, the blood is said to be alkalotic.

The respiratory buffer response

Carbon dioxide (CO_2) is a normal by-product of cellular metabolism. CO_2 is carried in blood to the lungs, which removes the same by controlling breathing. So, partial pressure of CO_2 in arterial blood ($paCO_2$) is determined by alveolar ventilation.

The excess CO₂ combines with water to form carbonic acid. The blood pH changes according to the amount of this acid in the body and so does the depth and rate of ventilation. Hence CO₂ is looked upon as a respiratory acid. [3]

- As blood pH decreases (acidosis), CO₂ is exhaled (alkalosis as compensation).
- As blood pH increases (alkalosis), CO₂ is retained (acidosis as compensation).

The respiratory response is fast and activated within minutes.^[3]

The renal buffer response

The kidneys secrete Hydrogen ion (H⁺) and reabsorbs bicarbonate. This is adjusted by the kidneys in response to metabolic acid formation.

Bicarbonate is a metabolic component and considered a base.

- As blood pH decreases (acidosis), the body retains bicarbonate (a base).
- As blood pH rises (alkalosis), the body excretes bicarbonate (a base) in urine.

This compensation is slow and takes hours to days to get activated. [3]

The acid-base control

The pH is dependent on the paCO₂/HCO³⁻ (HCO³⁻: bicarbonate) ratio.

A change in CO₂ is thus compensated by a change in HCO³⁻ and vice versa.

The initial change is called the primary disorder (e.g., change in CO₂ in this case).

The secondary response is called the compensatory disorder (e.g., change in HCO³⁻ in this case).

Basic facts to remember

- CO₂ is a respiratory component and considered a respiratory acid. It moves opposite to the direction of pH and is visualized as a see-saw [Figure 1] (as paCO₂ in blood increases, pH decreases—respiratory acidosis)
- 2. Bicarbonate is a metabolic component and considered a base. It moves in the same direction

- as pH and is visualized as an elevator [Figure 2] (as bicarbonate in blood increases, pH increases—metabolic alkalosis)
- 3. If CO₂ and HCO³⁻ move in the same direction, it is considered a primary disorder; for example, if there is respiratory acidosis in body (CO₂ retention), the bicarbonate levels increase as a compensation (metabolic alkalosis). The direction of both CO₂ and HCO³⁻ are the same in this case
- 4. If CO₂ and HCO³ move in opposite directions, it is considered a mixed disorder; for example, mixed disorder in the case of salicylate poisoning: Primary respiratory alkalosis due to salicylate-induced hyperventilation and a primary metabolic acidosis due to salicylate toxicity.

Conditions causing acid-base imbalance [Table 1][3]

Respiratory acidosis

It occurs due to any condition causing the accumulation of CO₂ in the body.

- Central nervous system (CNS) depression due to head injury
- Sedation (e.g., narcotics, postoperative, sedation), coma
- Chest wall injury, flail chest
- Respiratory obstruction/foreign body.

Respiratory alkalosis

It occurs due to decrease in CO₂. Here, hyperventilation occurs and CO₂ is washed out causing alkalosis.

- Psychological: Anxiety, fear
- Pain
- Fever, sepsis, pregnancy, severe anemia.

Metabolic acidosis (decrease in HCO³⁻)

It is caused due to excess of acids or deficit of base.

Increased acids

- Lactic acidosis (shock, hemorrhage, sepsis)
- Diabetic ketoacidosis
- Renal failure
- Deficit of base
- Severe diarrhoea and intestinal fistulas.

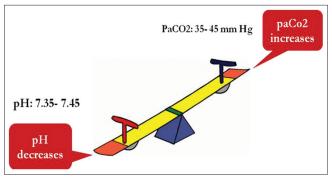


Figure 1: Visualization of pH and paCO₂ as a see-saw



Figure 2: Visualization of pH and bicarbonate as an elevator

*Metabolic alkalosis (excess of HCO*³⁻*)*

It is caused by excess base or deficit of acids.

- Acid Deficit: Prolonged vomiting, nasogastric suction, diuretics
- Excess base: Excess consumption of diuretics and antacids, massive blood transfusion (citrate metabolized to bicarbonate).

Why do we order a blood gas analysis?

- Aids in establishing diagnosis
- Guides treatmentplan
- Improvement in the management of acid/base; allows for optimal function of medications
- Acid/base status may alter levels of electrolytes critical to the status of a patient.

Limitations of blood gas analysis^[4]

- The blood gas analysis cannot yield a specific diagnosis. A patient with asthma may have similar values to another patient with pneumonia^[4]
- The analysis does not reflect the degree to which an abnormality actually affects a patient^[4]
- Blood gas analysis cannot be used as a screening test for early pulmonary disease.^[4]

Arterial versus venous blood gas analysis

It is traditional to draw arterial blood for paO₂, paCO₂, and pH measurements. It is the best indicator of how well the lungs are oxygenating.^[3] However, if the venous sample is obtained, it is recommended that the values be compared and interpreted keeping in consideration the given table [Table 2].

The venous blood gas report can be of significance in hemodynamically^[3] unstable patients and should not be discarded.

Obtaining an arterial sample

The order of preference is radial artery > brachial artery > femoral artery.

The radial artery is preferred due to ease of palpation, access, and good collateral supply.^[1]

The collateral supply to the hand is confirmed by the modified Allen's test [Figure 3].^[5]

Table 1: Components and normal values			
H+ pH	Hydrogen ions, inversely proportional to pH Acidity/alkalinity	35-45 mmol/L 7.35-7.45	
paO ₂ SaO ₂	Partial pressure of oxygen in arterial blood Arterial oxygen saturation	80-100 mmHg 95-100%	
paCO ₂ HCO ^{3.}	Partial pressure of CO ₂ in arterial blood Bicarbonate in blood	35-45 mm Hg 22-26 mEq/L	
BE	Base excess (amount of excess or	-2 to+2	
	insufficient amount of base in blood) -ve in acidosis, +ve in alkalosis	mmol/L	

CO₂: Carbon dioxide

Modified Allen's test^[5]

- Ask the patient to make a tight fist.
- Using the middle and index fingers of both hands, apply pressure to the wrist. Compress the radial and ulnar arteries at the same time (never use the thumb to detect the artery).
- While maintaining pressure, ask the patient to open the hand slowly. Lower the hand and release pressure on the ulnar artery only.
- Positive test: The hand flushes pink or returns to normal color within 15 seconds
- Negative test: The hand does not flush pink or return to normal color within 15 seconds, indicating a disruption of blood flow from the ulnar artery to the hand
- If the Allen's test is negative, the radial artery should not be used.

Sampling^[1]

- The arm of the patient is placed palm up on a flat surface, with the wrist dorsiflexed at 45°
- The puncture site should be cleaned with alcohol or iodine (allow the alcohol to dry before puncture, as the alcohol can cause arteriospasm), and a local anesthetic (such as 2% lignocaine) should be infiltrated

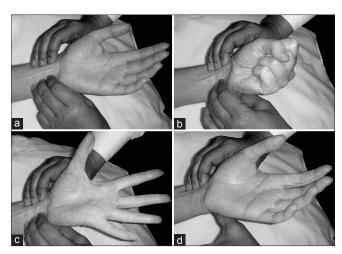


Figure 3: The modified Allen's test]: (a) The radial and ulnar arteries are palpated. (b) The patient clenches the fist and both the arteries are compressed manually. (c) The patient opens the fist (note the blanching of the palm). (d) The ulnar artery pressure is released; if the colour returns to pink, the test is positive; if the colour does not return to pink, the test is negative.

Table 2: Arterial versus venous blood gas				
Value	Arterial blood	Mixed venous		
pН	7.40 (7.35-7.45)	7.36 (7.31-7.41)		
paO ₂	80-100 mmHg	35-40 mmHg		
O ₂ saturation	95%	70-75%		
PaCO,	35-45 mmHg	41-51 mmHg		
HCO ^{3.[±]}	22-26 mEqL ⁻¹	22-26 mEqL ⁻¹		
BE	-2 to + 2	-2 to + 2		

(Table adapted from $^{(3)}$); O_2 : Oxygen, pa O_2 : Partial pressure of oxygen in arterial blood, pH: Acidity/alkalinity, PaCO $_2$: Partial pressure of oxygen in arterial blood, HCO 3 : Bicarbonate in blood, BE: Base excess

- The radial artery should be palpated for a pulse, and a preheparinised syringe with a 23- or 25-gauge needle should be inserted at an angle just distal to the palpated pulse [Figure 4]
- After the puncture, sterile gauze should be placed firmly over the site and direct pressure applied for several minutes to obtain hemostasis.

Errors[7]

- Allow a steady state after initiation or change in oxygen therapy, before obtaining a sample (in the patients without overt pulmonary disease, a steady state is reached between 3 and 10 minutes^[8,9] and in patients with chronic airway obstruction, it takes about 20-30 minutes)^[10]
- Always note the percentage of inspired air (FiO₂) and condition of the patient
- Flush the syringe with heparin or use preheparinised syringes. Do not use excess heparin as it causes sample dilution. Excess of heparin may affect the pH. Only 0.05 mL is required to anticoagulate 1 mL of blood. Because dead space volume of a standard 5 mL syringe with 1" 22-gauge needle is 0.02 mL, filling the dead space of the syringe with heparin provides sufficient volume to anticoagulate a 4 mL blood sample [10]
- Avoid air bubbles in syringe^[4]
- Avoid delay in sample processing. As blood is a living tissue, O₂ is being consumed and CO₂ is produced in the blood sample. The delay may affect the blood gas values. In case of delay, the sample should be placed in ice and such iced samples can be processed for up to two hours without affecting the blood gas values. [10]
- Accidental venous sampling. The venous sample report should not be discarded and can provide sufficient information.^[7]

Steps of interpretation

Step 1: Anticipate the disorder (keeping in mind the clinical settings and the condition of the patient) (e.g., the patient may present with a history of insulin-dependent diabetes mellitus (IDDM), which may contribute to a metabolic acidosis^[2]).



Figure 4: Radial artery sampling

Step 2: Check the pH.

If pH < 7.35: Acidosis

pH > 7.45: Alkalosis

pH = 7.40: Normal/mixed disorder/fully compensated disorder

(Note: If mixed disorder, pH indicates stronger component)

Step 3: Check SaO₂/paO₂ (SaO₂ is a more reliable indicator as it depicts the saturation of hemoglobin in arterial blood) Table 3.

Note: Always compare the SaO₂ with FiO₂, as the SaO₂ could be within normal range but still much less than FiO₂ if the patient is on supplemental oxygen (difference should be less than 10).

Step 4: Check CO₂ and HCO³⁻ (bicarbonate) levels – identify the culprit [Table 4].

Is it a respiratory/metabolic/mixed disorder?

Step 5: Check base excess (BE).

It is defined as amount of base required to return the pH to a normal range.

If it is positive, the metabolic picture is of alkalosis. If it is negative, the metabolic picture is of acidosis.

Either of bicarbonate ions/base excess can be used to interpret metabolic acidosis/alkalosis.^[7]

The following tables show the interpretation of arterial blood gas report on the basis of using BE as a metabolic index [Figures 5 and 6].

(Tables adapted from[11])

Step 6: Check for compensation.

Is there a compensatory response with respect to the primary change?

If yes: Compensated, if no: Uncompensated.

In case of compensation, does it bring the pH to a normal range?

If yes: Fully compensated, if no: Partially compensated. Example: If pH is 7.21, HCO³⁻ is 14, and CO₂ is 40.

It is a case of metabolic acidosis (as CO₂ is normal,

Table 3: Grading of hypoxemia			
	SaO ₂ %	paO ₂	
Mild hypoxemia	90-94	60-79 mmHg	
Moderate hypoxemia	75-89	40-59 mmHg	
Severe hypoxemia	< 75	<40 mmHg	

SaO₂: Arterial oxygen saturation

Table 4: Acid-base disorders	
Increased CO ₂ (>40 mmHg) Decreased CO ₂ (<40 mmHg) Increased HCO ³⁻ (>24 mEq/L) Decreased HCO ³⁻ (<24 mEq/L)	Respiratory acidosis Respiratory alkalosis Metabolic alkalosis Metabolic acidosis

HCO³⁻ is decreased). Expected compensation would be a decrease in CO₂ causing respiratory alkalosis. Now consider this table [Table 5]:

Rule of thumb

• To check the authenticity of a laboratory arterial blood gas report^[4,12]

$$H^{+} = 24 \times \frac{PCO_2}{HCO^{3-}} \tag{1}$$

Calculate this value from the arterial blood gas report

and if this value is equal to the H⁺ in the report, the arterial blood gas report is authentic.

Alternatively, subtract the last two digits of the pH (e.g., 20 in pH 7.20) from 80; this value is

Table 5: Expected compensation				
HCO ³⁻	pCO ₂	Compensation		
14	40	Uncompensated		
14 14	30 ↓ 20 ↓↓	Partially compensated Fully compensated		
	HCO ³⁻ 14 14	HCO³- pCO₂ 14 40 14 30 ↓		

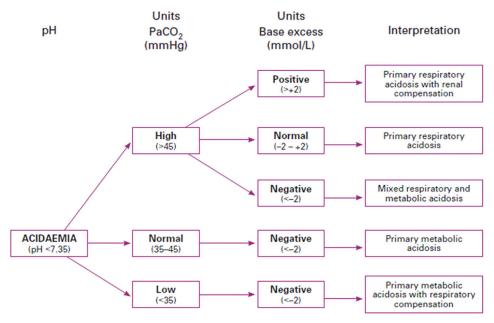


Figure 5: Interpreting acidaemia on an arterial blood gas result

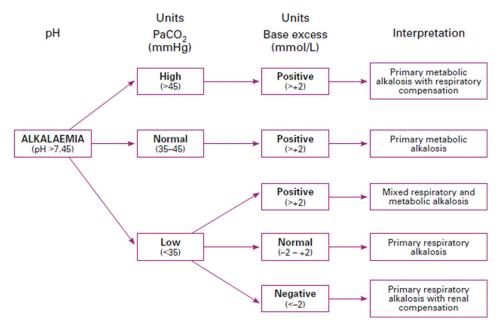


Figure 6: Interpreting alkalaemia on an arterial blood gas result

approximately equal to the H^+ concentration (proposed by Burden *et al.*^[13]).

For example, consider this arterial blood gas report: pH: 7.42, pCO₂:30.8, HCO³: 19.3, H⁺: 38.1.

Now, $H^+ = 24 \times \frac{30.8}{19.3} = 38.3 =$ approximately equal to measured H^+ in the report.

Alternatively, 80 - last 2 digits of pH = 80-42 = 38 = approximately equal to measured H + in the report.

So, the given ABG report is authentic.

Examples:

- 1. pH: 7.55, paCO₂: 49.0, HCO³: 48.2 The pH is alkalotic, paCO₂ is increased (retention of CO₂ causes acidosis), HCO³ is increased (increased base causes alkalosis). So, the primary disorder is metabolic alkalosis. Though CO₂ is being retained to compensate for the same, the pH has still not returned to a normal range. So, the interpretation would be partially compensated metabolic alkalosis.
- 2. pH: 7.34, paCO₂: 40.3, HCO³: 20.4 The pH is acidic, paCO₂ is normal, and bicarbonate is decreased. The primary disorder is metabolic acidosis, but there is no compensatory response as the paCO₂ is normal. So, the interpretation would be uncompensated metabolic acidosis.
- 3. pH: 7.52, paCO₂: 31.0, HCO³: 29.4 The pH is alkalotic, paCO₂ is decreased (alkalosis), and bicarbonate is increased (alkalosis).

As the directions of $paCO_2$ and bicarbonate are opposite and both are causing alkalosis, the picture is suggestive of a mixed disorder. The interpretation would be combined alkalosis.

CONCLUSION

Arterial blood gas analysis is a useful tool for diagnosis

monitoring and as an aid in management, but its value is limited by the understanding of the basic physiology and ability of the surgeon to interpret the report. Using a systematic and logical approach by using these steps would make the interpretation simple and easy to use.

REFERENCES

- Verma AK, Paul R. The interpretation of Arterial Blood Gases. Australian Prescriber 2010;33:124-9.
- Mary-Lynn Watson. Back to basics Acid Base Disorders. Can J CME 2002;6:57-63.
- Rao MS, Nagendranath V. Arterial blood gas monitoring. Indian J Anaesth 2002;46:289-97.
- Sood P, Paul G, Puri S. Interpretation of arterial blood gas. Indian J Crit Care Med 2010;14:57-64.
- Williams AJ. ABC of oxygen: Assessing and interpreting arterial blood gases and acid-base balance. BMJ 1998;317:1213-6.
- Asif M, Sarkar PK. Three-Digit Allen's Test. Ann Thorac Surg 2007:84:686-7.
- Barthwal MS. Analysis of arterial blood gases-A comprehensive approach. J Assoc Physicians India 2004;52:573-7.
- Mathews PJ. The validity of paO₂ values 3, 6 and 9 minutes after an FiO₂ change in mechanically ventilated heart surgery patients. Respir Care 1987;32:1029-34.
- Hess D. Good C, Didyoung R. The validity of assessing arterial blood gases 10 minutes after an FiO2, change in mechanically ventilated patients without chronic pulmonary, disease. Respir Care 1985;30:1037-41.
- Woolf CR. Letter: Arterial blood gas levels after oxygen therapy. Chest 1976;69:808-9.
- Drage S, Wilkinson D. Acid base balance. Update 13. 2001. World Federation of Societies of Anaesthesiologists. Available from: http://update.anaesthesiologists.org/wp-contentuploads/2009/09/ Acid-Base-Balance-Update-13.pdf [Last accessed on 2010 Jul 7].
- Kassirer JP, Bleich HL. Rapid estimation of plasma CO2 from pH and total CO2 content. N Engl J Med 1965;272:1067-8.
- Burden RJ, McQuillan PJ. Converting pH and H⁺: A "rule of thumb". Br J Anaesth 1997;78:479.

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